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support the statement that coronary artery disease mortality has been linked to a high-cholesterol diet.

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#### REFERENCES

- 1. Desmond S: Diet and cancer—Should we change what we eat?—Medical Staff Conference, University of California, San Francisco. West J Med 1987; 146:73-78
- 2. Hill AB: The environment and disease: Association or causation? Proc R Soc Med 1965; 58:295-300
- 3. Lowering Blood Cholesterol to Prevent Heart Disease. Natl Inst Health Consensus Dev Conf Consensus Statement 1985; 5

## The Role of Irradiation

To the Editor: I would like to comment on the article "Subacute Leukoencephalopathy Complicating Acute Lymphoblastic Leukemia" in the February 1987 issue. Cranial irradiation was implicated as a probable cause of the patient's severe delayed leukoencephalopathy. However, in the case presentation, no mention of irradiation was made. We are not told when in the course of the illness the patient received radiation treatment, what area was treated or the total dose and daily fractionation. These factors are all very important in evaluating the contribution of irradiation to the patient's clinical course. Although I do not question the probable role of the irradiation in the patient's illness, I do feel that we in the medical community have a responsibility to discuss radiation effects in objective and specific terms, with reference to the large body of available information.

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## REFERENCE

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## **Drs Filloux and Townsend Respond**

TO THE EDITOR: Dr Cole is quite justified in inquiring about the details of craniospinal irradiation in this patient, as a single sentence providing this information was inadvertently omitted from the final draft of our manuscript. The patient received 2,360 rads of cranial irradiation in 12 fractions over a three-week period beginning three months after the discovery of malignant cells in the cerebrospinal fluid, and 1,800 rads to the spinal axis in ten fractions during the

same period. We regret this serious omission and thank Dr Cole for bringing it to our attention.

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# Incidence of Cough Associated With Captopril Therapy

TO THE EDITOR: In an article in the February issue, McNally¹ discusses the incidence and potential pathophysiology of cough occurring in association with captopril therapy. The current labeling for captopril lists cough (along with several other adverse reactions) as occurring "in about 0.5 to 2 percent of patients but did not appear at increased frequency compared to placebo or other treatments used in controlled trials" involving captopril.

The published data cited for the 5% to 15% incidence used by the author are biased by the fact that the frequencies reported describe limited patient groups for whom cough has been a problem—in fact, the author's personal experience would suggest an even higher incidence.

From a data base of approximately 12,000 patients who received captopril in controlled clinical trials, 105 (0.9%) had cough reported as an adverse reaction. The vagaries of the postmarketing experience preclude a reliable frequency determination of any adverse reaction, since both the reaction (numerator) and the population exposed to the drug (denominator) cannot be easily quantified with any degree of accuracy.

Cough occurring in association with angiotensin-converting enzyme inhibitors is a recognized clinical entity, <sup>2,3</sup> albeit poorly understood. The incidence appears to be less than 2% when large groups of patients are reviewed, and individual clinical experiences must be analyzed in this context.

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- 2. Webb D, Benjamin N, Collier J, et al: Enalapril-induced cough (Letter). Lancet 1986; 2:1094
  - 3. Inman WHW: Enalapril-induced cough (Letter). Lancet 1986; 2:1218